

C. G. PETER LÖWENHIELM

**ON BRIDGING VEIN DISRUPTION AND
ROTATIONAL CEREBRAL INJURIES DUE
TO HEAD IMPACT**

Lund 1977

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C.G. Peter Löwenhielm

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From the Department of Forensic Medicine and the
Division of Solid Mechanics, University of Lund, Sweden

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This dissertation is based on the following publications:

- I. Voigt, G.E., Löwenhielm, C.G.P. and Ljung, C.B.A.: Rotational cerebral injuries near the superior margin of the brain. Acta Neuropath. (Berl.) 39, 201-209 (1977)
- II. Voigt, G.E. and Löwenhielm, C.G.P.: "Gliding contusions des Grosshirns. H. Unfallheilk. 117, 329-335 (1974).
- III. Löwenhielm, C.G.P.: Dynamic properties of the parasagittal bridging veins. Z. Rechtsmedizin 74, 55-62 (1974)
- IV. Löwenhielm, C.G.P.: Strain tolerance of the vv. cerebri sup. (bridging veins) calculated from head-on collision tests with cadavers. Z. Rechtsmedizin 75, 131-144 (1974)
- V. Löwenhielm, C.G.P.: Tolerance levels for bridging vein disruption calculated with a mathematical model. To be published in J. Bioengineering.
- VI. Löwenhielm, C.G.P.: Mathematical simulation of gliding contusions. J. Biomechanics 8, 351-356 (1975).
- VII. Löwenhielm, C.G.P.: Dynamic strain tolerance of blood vessels at different post mortem conditions. To be published in J. Bioengineering.

In the text these papers will be referred to by their Roman numerals.



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INTRODUCTION

Head injury ranks the prime killer in motor vehicle accidents. Therefore it is natural that substantial efforts in biomechanical research have been focused on various head injury mechanisms. While ultimately enough information might be available to provide tolerance data for any impact condition, the present state of the art is limited to a few specific cases. For the acquisition of tolerance data a purely empirical approach has often been necessary since the connection to mechanical processes has not been properly understood. Investigation of the mechanical response to trauma, therefore is desired. The more thorough the understanding is of different injury mechanisms (not only of the brain but also of other tissues in the human body) the more solid will the basis be for the design of optimal safety devices.

There are three different types of injuries caused by blows to the head. Whiplash injuries involve acceleration of the head when there is no direct contact with the impactor. Bullets and high velocity fragments can pierce the skull and the brain matter and thus produce penetrating injuries. However, the most common injuries are the nonpenetrating injuries caused by blunt trauma to the head. The biological response to head impact may involve: 1) concussion, defined as a reversible or irreversible disorder of brain function; 2) brain lesions, defined as visible structural damage and 3) skull fractures. Brain lesions can be primary and secondary (Unterharnscheidt and Sellier (1965)). Primary injuries originate in direct connection to the head impact and are focal or multilocular. The primary lesions induce secondary lesions which appear as edema, hemorrhages, partial or total necrosis.

Impact to the head gives rise to a motion that can be resolved into two components, translation or rotation (or linear or angular movement). The relative contribution of these components to head injuries has not yet been sufficiently clarified.

Rotational motion which results in shearing of tissues can be caused by whiplash or direct head impact. The original work in this area was presented by Holbourn (1943) who argued that the translation was an insignificant cause of brain damage. This view was later on supported by experimental work on animals carried out by Ommaya, Hirsch and Martinez (1966). They demonstrated that a cervical collar, which permitted only translation, protected the brain during impact. The results indicated that rotation was necessary to cause brain lesions at interesting levels of impact. Unterharnscheidt and Higgins (1969) reported that rotation gives rise not only to brain lesions but also to spinal cord injuries. Ljung (1975) by using mathematical simulation, showed that deformation of cm-magnitude arises inside the brain at sudden rotation of a magnitude that is frequently experienced in, for instance, traffic accidents.

The opinion that translational head motion is insignificant as a cause of brain injuries is not undisputed. Some workers have produced experimental data suggesting that pressure gradients caused by translation of the skull cause intracranial "cavitation". As a result cerebral concussion as well as cerebral contusion should arise (Unterharnscheidt and Sellier, 1966, Hodgson, 1970). Goldsmith (1966) claimed that cavitation is the result of rarefaction waves reflected from the skull boundary opposite from the side of the blow.

Ever since Gross (1958) succeeded to produce cavitation in water filled glass skulls subjected to impact, the cavitation theory for brain lesions has received much interest. The location of the so called coup and contre-coup lesions can be readily explained by this injury hypothesis. However, no experi-

mental results exist where lesions in brain matter can be obviously ascribed to cavitation.

In reality, one type of acceleration rarely occurs without the presence of the other type. Both excentricities of the impact and actions of the support system (the neck) account for this fact. Reconstruction of the course of events in motor vehicle accidents indicates that rotational acceleration occurs when unrestrained car occupants hit the steering-wheel, the dash-board or the wind-shield. Under these circumstances certain intracranial injuries are frequently found: a) bleedings near the superior margin of the cerebral hemispheres, situated in the cortex or in the subcortical white matter (rotational cerebral injuries (RC-injuries)), and b) disruption of the parasagittal bridging veins (vv. ccrebri sup.) (Voigt, Saldeen, 1968). Concomittant primary brain stem lesions and accute brain swelling seem to be the cause of death in these cases. Lindenberg and Freytag (1960) designated the subcortical bleedings as "gliding contusions". They assumed in agreement with Krauland (1949) that they resulted from stretching of the parasagittal bridging veins and the surrounding Pacchionian granulations. The stretching was considered to arise as a result of a sudden dislocation of the brain surface in relation to the inner side of the skull. Such dislocations were experimentally shown by Pudenz and Shelden (1946) and by Ommaya (1966). The same mechanism was also made responsible for the disruption of the parasagittal bridging veins (Unterharnscheidt and Sellier, 1971; Voigt and Saldeen, 1968). Information on the frequency and the late effects of these injuries however, has so far been lacking.

In order to establish tolerance levels for head injury physical and theoretical models, cadaver tests and in-vivo animal experiments must be utilized since in-vivo human tolerance tests cannot be performed and never will be. When cadaver test specimens are used it is of crucial importance to know how the mechanical properties of the different tissues change post mortem so that

accurate in-vivo tolerance levels can be established. This problem is avoided in in-vivo animal experiments, but then geometrical and scaling considerations must be taken.

Tolerance levels can be expressed in terms of known mechanical inputs to the head, e.g. acceleration, velocity, force and force duration. These parameters are then correlated to critical strains and stresses for the different tissues of the head. The duration of an impact has physical as well as physiological significance. Holbourn (1943) stated that long duration blows (compared with the natural period of the system) result in head displacements being proportional to the peak input acceleration, while for short duration blows, the displacements are proportional to the velocity change. Also von Gierke (1966) showed that the tolerance to trauma is dependent upon the mechanical frequency resonance of the respective tissue.

In order to correlate head impact levels to the injury degree several head injury criteria have been formulated. The Wayne State tolerance curve (Gurdjian et.al., 1964) was a phenomenological approach regarding simple linear skull fracture that often accompanies concussion. The parameters used were the linear acceleration of the head and the duration of the blow. Ommaya and Hirsch (1971) have published rotational injury tolerance criteria for cerebral concussion based on experiments with primates. The tolerance levels were given in terms of rotational velocity and rotational acceleration. Tolerance criteria for the frequently occurring bridging vein disruptions and RC-injuries have so far been lacking.

Against the background outlined the aim of the present study may be stated as follows:

- to study the epidemiology of bridging vein disruption and RC-injuries;
- to study the pathological-anatomical picture of RC-injuries with regard to different survival times;
- to investigate the dynamic properties of human bridging veins;
- to establish tolerance levels for bridging vein disruption;
- to study the genesis of the gliding contusions;
- to establish tolerance levels for gliding contusions; and
- to study post mortem changes of mechanical properties of blood vessels.

The working hypothesis is that the RC-injuries and bridging vein disruptions are caused by rotational movement of the head.

METHODOLOGICAL CONSIDERATIONS

Some of the methods and techniques used in this work are not included in the papers and are therefore presented here. Also, a brief presentation of the high-speed film analysis technique used is given.

Accident reconstruction

Most of the traffic accidents in papers I and II have been investigated by reconstruction of the course of events. At autopsy the external injuries to the head and body of the diseased were registered. These findings were indicated on a simple dummy, the size of which could be adjusted to the size of the diseased. By placing the dummy in the crashed car, the injuries could easily be correlated to the damage to the car, e.g. indentation of the dashboard, or a "star" in the wind-shield. Blood stains, skin and hair fragments found in the crashed car also indicated contact points as well as flakes of varnish and glass splinters in the wounds. These correlation measures allow reconstruction of the direction of force action and of the pattern of movement of the head and the body.

It was revealed that the head often is subjected to violent rotational movement. This occurs especially when the head strikes interior parts of the car such as the wind-shield, the dashboard or the steering-wheel. The most common situation giving rise to disruption of parasagittal bridging veins or RC-injuries, is impact to the head from in front or from behind.

Brain autopsy procedure

At autopsy the brain was removed ad modum Flechsig, i.e. the upper part of the cerebrum is cut off together with the roof of

the skull and then removed in block together with the dura by using a spatula. This procedure allows examination of the parasagittal bridging veins by carefully folding up the dura.

Histologic staining methods

Apart from standard brain staining methods (hematoxylin-eosin, van Gieson, Luxol Fast Blue and in some cases myelin staining according to Mahon), the neurofibrils and axons were impregnated by a new method which is a modification of Naoumenko and Feigin's method (1967).

1. Fix the tissues in 10 per cent formalin and embed in paraffin as usual. Cut at 6 to 10 microns, mount on glass slides, deparaffinize in xylol and rehydrate.
2. Place the slides in 10 per cent formalin.
3. Wash in 3 changes distilled water, alcohol, distilled water.
4. Place the section in the following solution for 24 hours at 56°C (Naoumenko and Feigin):
To 100 ml of buffered saline solution (1000 ml distilled water, 10 ml 0.1 per cent NaCl, 100 ml boric acid borax buffer, pH 7.6) add 3 ml 0.1 per cent silver nitrate solution.
5. Wash in 3 changes distilled water.
6. Place the section in the following solution for approximately 1 hour 15 min. and keep at 22-25° in the dark.
Add immediately before use 1 part of a fresh prepared solution consisting of 0.4 g hydrochinon, 1 g citric acid and 20 ml distilled water to 3 parts of a well shaken solution consisting of 40 ml 20 per cent aqueous arabic gum which is at least 14 days old and to which has been added some thymol crystals to guard against fungus and 0.4 ml 10 per cent aqueous silver nitrate. Shake well before using.
7. Wash in running tap water.
8. Wash 3 times in distilled water.

9. Place in 1 per cent aqueous gold chloride for 10 min.
10. Wash in distilled water.
11. Place in 1 per cent oxalic acid for 5 min.
12. Wash in distilled water.
13. Place in 5 per cent sodium thiosulfate for 2 min.
14. Wash, dehydrate, clear in methylbenzoate and mount permanently in Canada balsam solved in Xylene.

The method gives approximately the same results as that of Naoumenko and Feigin, but has in our hands proved more reliable. The essential difference between the two methods is that the present method uses the arabic gum as a protective colloid, retarding the reduction-oxidation reaction between the silver ions and the reducing hydroquinone, while in Naoumenko/Feigin's method this retardation is achieved by sodium sulfite while the tissue probably is acting as a protective colloid (cf. Liesegang, 1924).

Experimental test equipment

Tensile tests have been performed on bridging veins from man and cows, carotid arteries from rats and jugular veins from rats (papers IV and VII). For this purpose a tensile test device was designed and developed by which small blood vessels could be strained to disruption with various constant strain rates (cf. figs. 1-3). The test specimen was mounted between two clips or tied to two hooks, one of which was stationary and connected to a piezoelectric force transducer so that the force-time history could be recorded during the test. The other hook or clip was attached to a movable guided yoke which could be accelerated practically seen instantaneously to a constant velocity. This was achieved by means of pneumatic devices. Depending on the velocity range desired, three different methods were applied. For low velocities (0.015-1.5 m/sec) the yoke was pushed by a conventional pneumatic piston (cf. fig. 1). For medium velocities (1.5-5 m/sec) the pneumatic

piston was used as an impact cylinder which transferred its kinetic energy to a massive steel cylinder sliding in a tube. This cylinder, in turn, transferred its kinetic energy by central impact to the yoke, (cf. fig. 2). For high velocities (5-15 m/sec) the pneumatic cylinder was omitted and the tube closed at one end. Compressed air was used to accelerate the steel cylinder (cf. fig. 3). After passing a number of evacuation ports in the tube the cylinder was not appreciably affected by the driving pressure, and transferred its energy to the yoke.

The loss of velocity of the yoke during trial (primarily due to friction) was less than 4% and the test velocity was therefore considered constant.

The velocity of the yoke was measured by using two position markers Pencil leads incorporated as switches in an electric circuit (cf. figs. 1-3), mounted at a well defined distance apart. During trial they were broken by the yoke and the time lag between the breaks was measured.

In early tests the force transducer signal was recorded by an oscilloscope, while in a later version of the test equipment a transient recorder was used. The contents of the transient recorder could be loaded into a desktop programmable calculator for further processing, e.g. numerical filtering and printing of the results.

At high test velocities disturbing high frequency vibration occurred in the test equipment. The force signal was therefore low-pass filtered by using a 2-pole Butterworth active filter with a cut-off frequency of 5000 Hz and a damping of 6 dB/octave.

Figs. 1-3 Experimental equipment for tensile tests on small blood vessels. Appearance a) before and b) after a test. Fig. 1. Setup for low and medium velocities. Fig. 2. Setup for medium velocities and Fig. 3. Setup for high velocities.

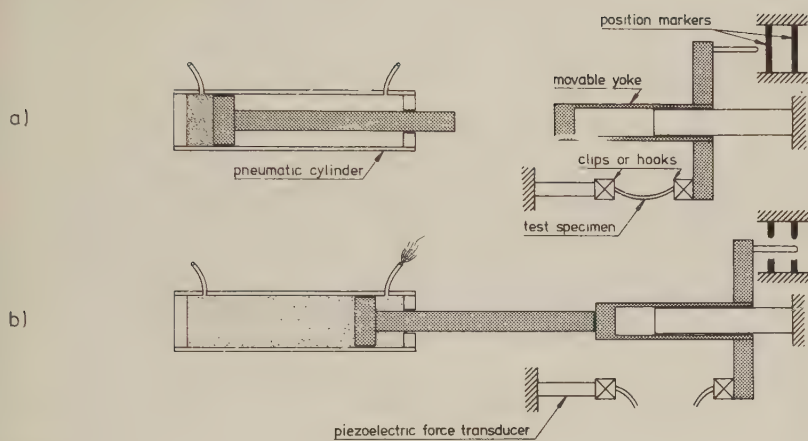


Fig. 1

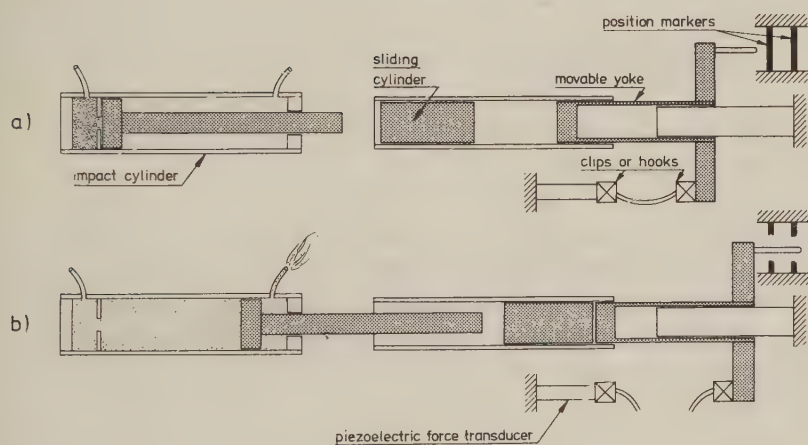


Fig. 2

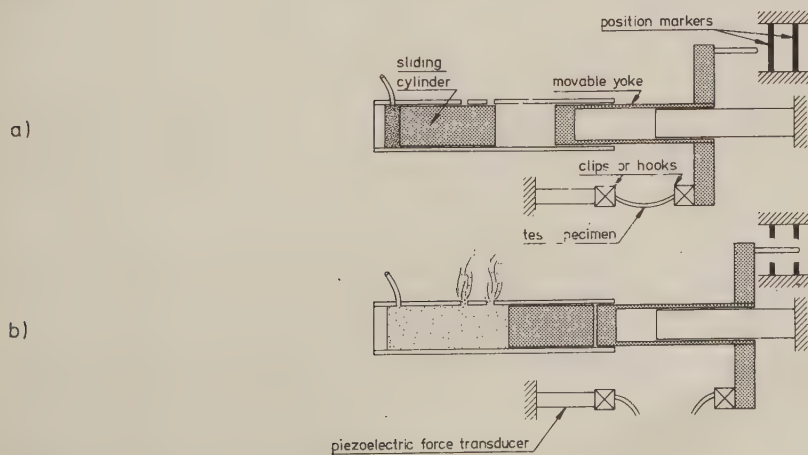


Fig. 3

Since the velocity of the yoke was regarded as constant the time scale in the force records could be directly used for obtaining the current length of the blood vessel. Thus both the strain and the strain rate could be computed. The strain rate then has been defined as

$$= \frac{\text{extended length} - \text{original length}}{\text{original length}}$$

and the strain rate as

$$= \frac{\text{impact speed}}{\text{original length}}$$

The original length corresponds to a completely stretched blood vessel at negligible load.

Recordings show that the force reaches a peak or a plateau and then returns gradually to the base line. The tissue components of the tested blood vessel are assumed to begin to disrupt when a plateau or the first peak of the force recording is reached. The length of the blood vessel at this point is therefore defined as the extended length.

One difficulty in the performance of tensile testing is to avoid boundary effects at the attachment points. Unsuitable connection to the test device weakens the rupture resistance. In spite of careful mounting of the blood vessels, disruption sometimes occurred at a connection site. To rule out possible influences of improper mounting these results were excluded in the further analysis.

Another factor that should be taken into consideration is the possible effects of wave propagation. Since the blood vessels tested were inserted between one fixed and one movable point it can be surmised that the portion of the bridging vein which is closest

to the movable point of insertion would be stretched and disrupted before the portion closest to the fixed point of insertion would be affected to any appreciable degree. Disregard of the effects of wave propagation is justified if a large number of wave reflections at the ends occur before disruption. About 5 reflections can be considered as sufficient. The average propagation velocity of longitudinal waves in vein tissue during the course of events can be estimated to about 50 m/s. A wave would be reflected more than 5 times in a 1 cm long blood vessel as soon as the time to disruption is longer than 1 msec. In the present experiments the loading time is always longer than 1 msec and the lengths of the blood vessels tested were shorter than 1 cm. Thus the effects of wave propagation can be safely disregarded.

Film analysis

In paper IV analyses of high speed films have been made. From the films the time-displacement history is obtained. In order to obtain velocities and accelerations, differentiation with respect to time has to be performed. For this purpose smoothed cubic spline functions have been used in order to obtain high quality derivatives (Berghaus and Cannon, 1973; Reinish, 1967). A spline is a piece-wise-interpolation polynomial defined over a set of points (t_i) . Individual cubic functions $h(t_i)$ are obtained for each interval (t_i, t_{i+1}) with the derivatives to the second order held continuous over the entire span (t_o, t_n) . The functions (t_i) may be allowed to deviate from the data which assures a smoother first and second derivative than if the functions are constrained to pass through all data. It has been assumed that the differentiation of high speed photography data could not be as accurate as integration of accelerometer data for the desired kinematic quantity. However, Calcote (1977) has shown that this is not the case, and he concludes that cine data will yield results that are at least as accurate as accelerometer data.

SUMMARIES OF THE PAPERS

Rotational cerebral injuries near the superior margin of the brain (I)

The most frequent traumatic injury around the superior margin of the brain is disruption of parasagittal bridging veins and/or cerebral lesions typically located to the posterior part of the superior frontal gyrus or to the central gyrus. In the fresh state the cerebral lesions are characterized by subarachnoidal bleedings, cortical and mainly subcortical perivascular bleedings. Lindenberg and Freytag, (1960) designated the subcortical bleedings as gliding contusions. These lesions often occur without skull fractures.

In 658 consecutive cases of blunt trauma the above mentioned lesions were searched for.

Disruption of parasagittal bridging veins was seen in 29% of the cases studied and cortical and subcortical bleedings close to the superior margin of the brain in 11%. In 19% of the cases with bridging vein disruption there was no skull fracture. The corresponding figure for the cortical and subcortical lesions was 25%.

When many or all of the bridging veins have been disrupted, death occurs immediately or later after persistent unconsciousness. Concomittant primary brain stem injuries (confirmed by the presence of small perivascular hemorrhages) are probably the direct cause of death. Most striking in these cases is the absence of a noteworthy subdural hematoma even if life has been maintained by respirator treatment. The brain shows acute swelling but without flattening of the gyri. The brain weight is 200-300 g above normal. There is usually a bilateral tentorial impression in the parahippocampal gyri often with surrounding cortical hemorrhages. Subarachnoidal

hemorrhages are observed at the upper margin of the brain. The characteristic subcortical bleedings are always rodshaped, usually occurring bilaterally and not continuous with the subarachnoidal hemorrhages. Additional small cortical hemorrhages which may be seen in the depth of the sulci in these cases cannot be the consequence of infarction since they are found in cases of very sudden death.

Longer survival time is possible if only a few or no bridging veins are disrupted. The subcortical bleedings are generally restricted to the perivascular space, but may after 2.5-3.5 h penetrate the surrounding brain tissue and assume considerable size.

If the subcortical bleedings remain limited to the perivascular space, the patient may survive, provided there are no other serious injuries. With time the perivascular bleeding becomes organized, leaving a perivascular scar of connective tissue. Iron pigment can be shown in the scar tissue for a long time afterwards, as is also the case in the meninges after subarachnoidal hemorrhage. In the surrounding tissue retraction balls of the axons may be demonstrable. Even with a lack of discernible cortical and subcortical bleedings small foci of pallor (myelin preparation) can be recognized after 2 weeks. Retraction balls of the axons can be demonstrated around these foci too. After longer survival time the subcortical lesions can be interpreted as scars after incomplete necrosis. Thus the trauma responsible for the subcortical bleedings causes lesions not only of the blood vessels, but of the cerebral tissue as well.

It is sometimes difficult to decide whether these lesions are the immediate result of head trauma or whether the hemorrhages and necrosis develop secondarily. After a thrombosis of the superior sagittal sinus cortical and subcortical hemorrhages with incomplete necrosis can be seen. Subdural hematomas between the falx cerebri and the medial side of a cerebral hemisphere may displace the hemisphere and give rise to additional bridging vein disruption.

Neurosurgical treatment of a bleeding parasagittal bridging vein can result in cortical and subcortical bleeding.

The cortical and subcortical bleedings (RC-injuries) should be distinguished from the classical cortical contusion which in the early state is characterized by local subarachnoidal hemorrhages and by pinpoint bleedings at the crests of the gyri and in the later stages by complete necrosis of the injured area. Cortical contusions located to the superior margin of the brain is seldom seen unless in association with a fracture of the roof of the skull.

"Gliding contusions" des Grosshirns (II)

This paper is related to I. However, special emphasis was made with regard to the RC-injuries in children. Findings, which are not included in I are mentioned here.

The material studied consisted of 641 consecutive autopsies where death had been caused by blunt trauma. The cases were grouped according to the age. The frequency of gliding contusions and of disruption of the parasagittal bridging veins were studied for each age group. The relative frequency of disruption of the bridging veins was significantly lower for persons with an age more than 40 years than for younger persons. The reason for this difference is thought to be an effect of connective tissue which anchors the soft cerebral membranes (covering the surface of the brain) to the dura along the superior sagittal sinus. In children the amount of connective tissue is sparse. With age the proliferating connective tissue finally constitutes a firm connection. Therefore, slip between the surface of the brain and the dura would be prevented and the tolerance to bridging vein disruption increased. On the other hand the deformation capabilities of the brain is unaffected by the connective tissue. This fact then explains that the gliding contusion/bridging vein disruption quotient is higher for older persons.

In the youngest age group (<11 years) disruption of the bridging veins occurred more frequently (50%) in comparison with the frequency of the total material (24%). In 12 of 13 cases in the youngest age group, numerous or all bridging veins were disrupted. Remarkable was also, that only 40% of the children below 11 years of age had skull fracture - the corresponding value for the total material was 77%.

Dynamic properties of the parasagittal bridging veins (III)

Disruption of parasagittal bridging veins generally occurs in the subdural space. Blunt trauma to the head causes displacements of the brain relative to the skull and so the bridging veins will be strained. At static loading the ultimate strain for various blood vessels is more than 100%. Since it was estimated that such large strains hardly occur in several cases where disruption of parasagittal bridging veins have been found, an investigation of the dynamic properties of such veins was undertaken. Dynamic tensile tests were performed by using special test equipment which is described on page 13.

The results indicate that the ultimate strain of the bridging veins is dependent on the strain rate, such that the ultimate strain decreases at increasing strain rate. The ultimate strain for strain rates exceeding $1000 \cdot \text{sec}^{-1}$ is about 20%, approximately a five-fold reduction from the static case.

Strain tolerance of the vv. cerebri sup. (bridging veins) calculated from head-on collision tests with cadavers (IV)

Voigt and Lange (1971) and Voigt et.al. (1973) simulated head-on collision experiments with unembalmed cadavers. In some of these experiments bridging vein disruption was produced. High speed films from these tests were analysed with respect to head angular velocity and acceleration. From the films the angular displacement

history of the head was obtained and in order to get high quality derivatives from these data, smoothed cubic-spline functions were used (see Methodological considerations). The experimental data are presented in terms of angular acceleration and angular velocity change. It is shown that the criterion for bridging vein disruption implies that both a critical angular acceleration and a critical angular velocity change shall be exceeded. The result indicates that the critical level regarding the maximal acceleration is about $\ddot{\phi} = 4500 \text{ rad/sec}^2$ (cf. fig. 4).

Unterharnscheidt and Higgins (1969) conducted carefully controlled studies on head rotation of squirrel monkeys. In their results the pathological-anatomical findings indicated bridging vein disruption in some cases. Scaling (cf. Ommaya, 1967), yielded an estimate of the critical angular velocity change to $\Delta\dot{\phi} = 50 \text{ rad/sec}$.

Tolerance levels for bridging vein disruption calculated with a mathematical model (V)

In this paper an attempt is made to validate the tolerance levels for bridging vein disruption proposed in paper IV. Bridging vein disruption is mostly seen in traffic casualties but can as well be demonstrated in persons who have fallen to the ground from an upright position. In such cases it is sometimes possible to make fairly accurate reconstructions of the accidents. Thereby the velocity part of the disruption criterion for bridging veins can be estimated.

In a recent case, disruption of bridging veins and brain stem injuries occurred as the result of a child's fall from a chair. Based on this case, which allowed thorough reconstruction, possible head angular acceleration and velocity were studied by means of a mathematical model. In this model the yielding properties of the impacted surface were taken into account. The head was regarded as a homogeneous sphere and the influence of the neck was neglected.

The expressions obtained for the angular movement of the head indicate that the angular displacement is quite small (4 deg.) during impact and that the maximal angular velocity is independent of the softness of other than very soft hit surfaces, thus essentially dependent only on the impact velocity.

The calculations suggest that the acceleration criterion proposed for bridging vein disruption (IV) may be correct while the velocity criterion seems to be somewhat too high. The velocity criterion previously proposed was derived from animal experiments. It is emphasized that factors such as differences in the geometrical shape of the skull-brain system of man and animal imply uncertainties when data from animal experiments are transferred to man.

This paper is based on a separate case but the result is supported by the fact that bridging vein disruption can often be found in cases where a person has fallen to the ground from a standing position. One conclusion of practical interest is that a moderate fall can give rise to disruption of parasagittal bridging veins regardless of the hit surface or object, unless it is very thick and soft. Such knowledge is necessary, for example in the judgement of suspected battered child cases.

Mathematical simulation of gliding contusions (VI)

Gliding contusions occur predominantly subcortically in the posterior part of the superior frontal gyrus and in the upper part of the anterior central convolution. They appear in the acute phase as streak-like perivascular bleedings apparently originating from intracerebral portions of the superior cerebral veins. Often the cerebral cortex is left intact. The pathological-anatomical picture and the location of this lesion, therefore suggest a causing

mechanism different from that of cortical contusions, which in the fresh state are characterized by pinpoint bleedings in the cerebral cortex at the top of the gyri and surrounding subarachnoidal hemorrhages.

This paper offers an explanation to the genesis of the gliding contusions and proposes tolerance levels in terms of critical head angular acceleration and critical head angular velocity change. For this purpose the deformation of the brain matter close to the superior sagittal sinus was simulated by means of a mathematical model. The rigid structures encasing the brain are the spherically shaped skull and the flat cerebral falx. Because of this geometry, the mathematical model could be simple (a Kelvin-Voigt viscoelastic material (brain) contained in a hollow hemispherical shell (skull) with a rigid wall (falx)), and yet offer an accurate study of the deformation in the interesting part of the brain.

The material parameters regarding the brain (shear modulus and dynamic viscosity) were determined from model tests on fresh cadaver brain, and regarding the blood vessels (ultimate strain as a function of the strain rate) from tensile tests on parasagittal bridging veins.

The model was subjected to a single angular acceleration pulse. The resulting brain deformation was expressed as the shear of the brain matter with respect to time. The result indicated that maximal shear of the brain matter is obtained at a certain distance below the surface of the brain. This distance is approximately 8 mm and corresponds well to the location of the gliding contusions. The shear levels in the cortex were found to be fairly low compared

to the levels obtained subcortically. This result is in good agreement with the fact that the cortex can be preserved uninjured while the deeper lying brain tissue can show extensive lesions.

In order to establish tolerance levels for gliding contusions, blood vessel disruption was chosen as the decisive criterion for injury. For this purpose the mathematical simulation was performed such that the angular accelerations and the angular velocity change were evaluated in the mathematical simulation. The critical values found for gliding contusions were $\ddot{\alpha}=4500 \text{ rad/sec}^2$ and $\dot{\alpha}=70 \text{ rad/sec}$ respectively (cf. fig. 4).

The deformation of the brain matter is of cm-magnitude when blood vessel disruption takes place. Such deformation implies considerable strain of the nervous tissue which consequently can be injured too.

Dynamic strain tolerance of blood vessels at different post mortem conditions (VII)

The mechanical properties of the different tissues of the human body are altered post mortem. In papers III and IV the results were obtained from investigations on cadaver bridging veins. In this paper a quantitative investigation on the post mortem mechanical changes of blood vessels is made in order to create a possibility to estimate the reliability of previously proposed tolerance levels regarding bridging vein disruption. For this purpose parasagittal bridging veins from cows, carotid arteries from rats and jugular veins from rats were tested with regard to the ultimate strain. The tests were carried out just after death and two days after death. The bridging veins from cows were kept in a wet chamber while the blood vessels from rats were kept either in situ or in a wet chamber.

The results indicate that at higher strain rates, the ultimate strain for bridging veins from cows is lower just after death than two days after death. This difference in ultimate strain is dependent on the strain rate and the difference increases at increasing strain rate. Regarding the blood vessels from rats, no significant difference from the results from tests just after death was obtained if the blood vessels had been kept in situ for two days after death. On the other hand a significant increase in ultimate strain was observed if the blood vessels had been kept in a wet chamber for two days.

The results suggest that the tolerance levels for bridging vein disruption proposed in paper IV might be affected. The acceleration criterion was obtained from cadaver tests. According to the results of this paper, a reduction of the acceleration tolerance level may be justified. Such a reduction however, does not need to be very substantial since the strain rates in this case are not very high and the post mortem change of the ultimate strain is not significant at low strain rates.

GENERAL DISCUSSION

In the investigation of pathogenesis, frequency, morphology and tolerance levels of bridging vein disruption and RC-injuries, different approaches have been used: Epidemiological and morphological studies (I, II) analysis of cadaver head-on collision tests (IV), establishment of intra-cranial tissue mechanical data (III) and simulation of the lesions by the use of mathematical models (V, VI). In addition an investigation of the postmortem changes of the material properties of blood vessels has been carried out in order to check the tolerance levels proposed for bridging vein disruption and for RC-injuries (VII).

The tolerance levels for bridging vein disruption proposed in paper IV were derived from head-on collision tests with cadavers and from scaling of results from in-vivo tests with squirrel monkeys. The cadaver tests yielded a critical angular acceleration of the head. However, it was felt that this value should be checked since experience from cadaver collision tests has shown more severe injuries than in corresponding real life situations. Experiments with living and dead pigs subjected to comparable collision situations (Löwenhielm et.al., 1977) could confirm this difference, at least concerning thoracic injuries. Paper VII reports on postmortem changes of the bridging veins from cows and neck vessels from rats. In some cases significant differences could be noted between tests carried out after two days compared to tests carried out just after death, but the differences found were dependent on the preservation method. However, the results indicated that the postmortem changes would effect the tolerance levels only to a slight degree.

The monkey experiments yielded a critical angular velocity change, by using a scaling procedure published by Ommaya et.al. (1967). It should be remarked that the scaling laws used are not quite incontrovertible, since viscosity effects are present. These effects are, however, probably not very important but some uncertainty prevails at such large scaling as from monkey to man. These problems are presently being investigated by Ljung (1977). In addition to the uncertainties regarding the scaling procedure, great care always has to be exercised when tolerance data from animals are transferred to man, among other reasons since the skull-brain geometry is different (Ward, 1977). In paper IV (discussion based on a child's fall from a chair resulting in bridging vein disruption) an attempt to check these data was made. The result indicated that the critical angular velocity change previously suggested should be reduced from 50 rad/sec to 30 rad/sec. No reason to change the suggested angular acceleration criterion was found. It must be emphasized that the results were based on a case regarding a child and that the incidence of bridging vein disruption is about twice as common in children compared to the adult (cf. II). Therefore, reduction of the velocity criterion for bridging veins is reasonable since it should include the child as well as the adult.

Tolerance levels for gliding contusions (VI) were calculated by mathematical simulation. The material properties used in the model were obtained from experiments on cadaver material. The significance of postmortem changes of blood vessels and brain matter must therefore be considered. As regards the blood vessels the ultimate strain data for bridging veins presented in paper III were used. These data have been validated with respect to postmortem changes in paper VII. In this paper, it is concluded that if any reduction should be made, the tolerance levels should be slightly reduced. The reason is that blood vessels, which have entered the mechanically stabilized state postmortem can be strained more than blood vessels in vivo (cf. VII). Concerning the material properties for the brain matter, data presented by Ljung (1975) were used. He used fresh unembalmed cadaver human brain which he subjected to rotational acceleration. Earlier investigations on this matter (Schuck and Advani, 1972) used continuous excitations of the brain matter.

In order to better simulate the conditions obtained when there is a sudden blow to the face or occiput, a single sudden rotational loading was chosen, and the deformation of the brain matter was recorded. By fitting a Kelvin-Voigt-rheological model to the deformation measurements the material properties were determined in terms of kinematic viscosity and shear modulus. It was found that the optimal set of parameters corresponded to a shallow minimum of the sum of the squared errors, i.e. the choice of material property data is not very critical. These data are the best ones available for simulation of gliding contusions. The potential uncertainty of this simulation therefore is, that the significance of the postmortem effects on the material properties of the brain tissue cannot be sufficiently established, since the in-vivo properties cannot be tested. The tolerance levels for gliding contusions obtained by mathematical simulation agree with practical experience concerning the relative frequency of bridging vein disruption and gliding contusions.

Sometimes gliding contusions occur without accompanying bridging vein disruption (cf. I). This finding seems to be contradicted by the fact that tolerance levels for gliding contusions are higher than those for bridging vein disruption. It has been remarked in paper II that the gliding contusion/bridging vein disruption quotient is higher for older persons than for young ones. This may be explained by proliferating connective tissue which with time will constitute a firmer attachment of the parasagittal surface of the brain to the dura. Thus slip is gradually prevented and the bridging veins are protected while deformation of the brain is unaffected. This seems to be the reason why gliding contusions can occur isolated. Another factor that should be remembered is that tolerance levels for gliding contusions are calculated for head impact from in front or from behind.

Aldman et.al. (1976) dropped different motor-cyclist helmets containing a dummy head onto a simulated moving road surface. Linear and angular accelerations of the dummy head were recorded. With a vertical impact velocity of 5.1 m/sec and a horizontal velocity, 8.3 m/sec of the road surface, angular accelerations ranging from 4500 rad/sec² to 14.500 rad/sec² were obtained. This experiment confirms the view that strong angular accelerations of the head occur in such cases. According to paper I bridging vein disruption and RC-injuries are overrepresented in motorcyclists compared to the total material studied. The results presented by Aldman et. al. (1976) give values which exceeded the critical angular acceleration proposed. The linear accelerations obtained were given as HIC-values and amounted to about 1000. The Head Injury Criterion (HIC) (Versace, 1971) is an interpretation of the Wayne State tolerance curve mentioned earlier, and a HIC-value of 1000 indicates that linear accelerations have attained a concussion level, i.e. functional brain disturbance but no visible pathological brain damage occurs. It should be pointed out that the basis of the Wayne State tolerance curve is linear acceleration data from impacts of heads of cadavers on surfaces of different softness. No attempt was made to estimate the angular mode of head motion (which is likely to occur in association with the impact (cf. VI)). The relative contribution of the rotational effects therefore cannot be established. In this context investigation on cerebral concussion carried out by Ommaya, Hirsch and Martinez (1966) should be commented. As mentioned in the introduction they used a cervical collar in order to prevent head angular acceleration in Rhesus monkeys subjected to head impacts. They found that the test animals could withstand a substantially higher linear acceleration than if a cervical collar was not used. This result immediately emphasizes the importance of taking the rotational movement of the head into consideration when tolerance levels for head injury is established. In other investigations by Ommaya and

Hirsch (1971) different primates (squirrel monkeys, rhesus monkeys and chimpanzees) were subjected to carefully controlled head angular accelerations. Tolerance levels for cerebral concussions were established and by scaling of these data the tolerance level for cerebral concussion in man was found to be about 1800 rad/sec^2 . Thus, the mechanism of cerebral concussion can consist of head rotation solely.

It has for some time been known that rotational movement of the head causes deformation and dislocation of the intracranial contents (Pudenz and Shelden, 1941; Holbourn, 1943). Ljung (1975) made an extensive mapping of the brain deformation at the typical site of bridging vein disruptions and RC-injuries. He showed that the deformation of the brain matter can attain cm-magnitude when the head is subjected to a sudden rotation representative for a car occupant in head-on collision.

The location of bridging vein disruption and RC-injuries is consistent with explanations of a causing mechanism resulting from the angular motion of the head. It appears to be very difficult to find this location compatible with arguments based on the translational motion. Translation of the head gives rise to pressure gradients within the brain but only to completely negligible displacements. If the pressure by some mechanism (cavitation, rarefaction of pressure waves) should produce intracranial lesions, these should occur where the pressure is low or high, i.e. near the impact pole of the brain ("coup") or near the remote pole of the brain ("contre-coup"). However, the pressure at the usual site of bridging vein disruptions or of RC-injuries is neither very high nor very low, why such a causing mechanism seems unlikely. Cavitation has been shown experimentally in waterfilled glass skulls (Gross, 1958) but never in brain matter. Stålhammar (1974) subjected in-vivo rabbit brain to subatmospheric pressures. No morphological changes, which could be attributed to pressure effects, were produced.

Response measures of a three-dimensional finite element brain model were compared to experimentally produced brain displacements by Ward (1977). In one version of the model slip along the skull-brain interface was allowed. Model simulation could reproduce the motion of the brain surface obtained in the lucite-calvarium tests carried out by Pudenz and Shelden (1943). These results confirm the opinion that bridging vein disruption is caused by strain rather than by some cavitation effect.

In experiments carried out at the University of California at San Diego (briefly reported by Ward (1977)), no measurable slip between the brain and skull was obtained during head impact. Alter et.al. (1976) subjected live monkeys to rotational sinusoidal loading with a frequency ranging from 1 to 35 Hz. Some of their results are briefly reported by Ward (1977). A cinefluorographic system was used to register the displacement of radiopaque spheres of hollow lead glass inserted 10 mm into the brain close to the midline. No measurable displacements could be observed. Because of these findings the brain model by Ward (1977) was revised. Slip along the skull-brain interface was eliminated. By using the above mentioned excitation, model simulation yielded brain displacements, which near the brain surface was negligible with respect to the skull. These results seem to contradict the causing mechanism proposed for gliding contusions (Vl). However, attention must be called for as the excitation used produced a peripheral acceleration of the skull of less 3 g or less. This value must be considered as low compared to 45 g, i.e. the value corresponding to the tolerance level for gliding contusions. Furthermore, the tolerance level for gliding contusions is still higher in monkeys because of scaling effects. A monkey brain-weight of about 50 grams would imply a three-fold increase to 135 g of the tolerance level (cf. Ommaya et.al. (1967)). The failure to detect intracranial motion in the experiments by Alter et.al. (1976) can also be explained by using the data for brain matter obtained by Ljung (1975). Simple calculations then reveal that the displacement amplitude expected is indeed very small, of the order of a tenth of a millimeter or less.

RC-injuries and bridging vein disruption are, of course, not the only kinds of intracranial injuries associated with head impact. A rotational acceleration of the head could hardly leave the caudal part of the brain unaffected. Here, too, considerable strains must be expected, particularly in the connection between the cerebrum and spinal cord. Thus it is not surprising that a rotational acceleration of the head (especially about a transverse axis), in addition to bridging vein disruption and RC-injuries frequently gives rise to brain stem injuries, often leading to immediate death. This seems to be the reason why such cases have not attained great clinical interest since the patients are already dead on admission to hospital.

In view of the preceding discussion the dominating role of the head angular motion as a cause to the intracranial injuries studied in the present work, seems to be solidly based. The resulting intracranial motion is effective at the locations where RC-injuries and bridging vein disruption occur. The displacement amplitudes are large enough to explain these injuries by simple mechanical considerations based on experimentally found data and estimates on the strength of the various tissues involved.

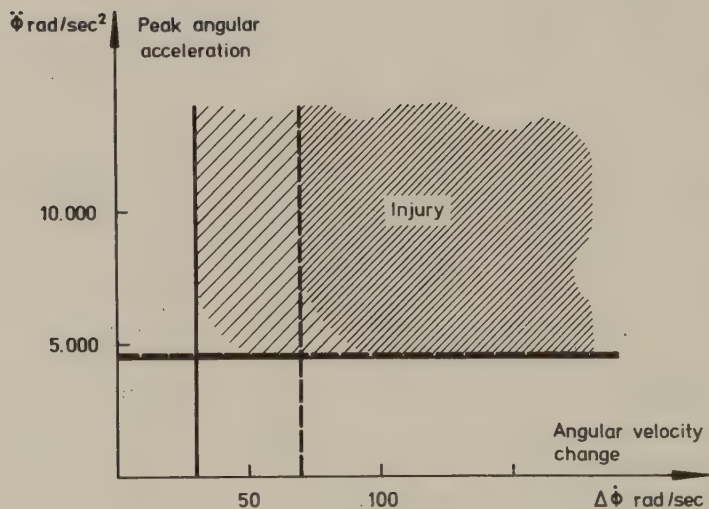


Fig. 4. Tolerance levels for bridging vein disruption (————) and for gliding contusions (————). It should be remarked that close to the intersection between the lines of critical acceleration and critical velocity change the tolerance levels can be slightly exceeded as indicated by the hatched areas.

CONCLUSIONS

- . In trauma cases, bridging vein disruption and RC-injuries are common intracranial lesions close to the superior margin of the brain. In consecutive postmortem examinations where death had been caused by blunt trauma to the head, bridging vein disruption was demonstrated in 29% and RC-injuries in 13% of the cases.
- . In addition to bridging vein disruption and/or RC-injuries, primary brain stem injury can frequently be demonstrated.
- . Bridging vein disruption and RC-injuries are explained by a sudden head rotation, which gives rise to slip along the brain-skull interface and to deformation of the brain matter.
- . The tolerance criterion for bridging vein disruption is that both a critical angular acceleration ($\ddot{\phi} = 4500 \text{ rad/sec}^2$) and a critical angular velocity change ($\Delta\dot{\phi} = 30 \text{ rad/sec}$) shall be exceeded.
- . The tolerance criterion for subcortical RC-injuries (gliding contusions) is that both a critical acceleration ($\ddot{\phi} = 4500 \text{ rad/sec}^2$) and a critical angular velocity change ($\Delta\dot{\phi} = 60 \text{ rad/sec}$) shall be exceeded.

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